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What Is Your Neurologic Diagnosis?

An 8-year-old 6.5-kg (14.3-lb) neutered male Chinchilla-cross cat was referred to the Neurology Service at the School of Veterinary Sciences, University of Bristol, 48 hours after sudden-onset paraparesis. Initially, the cat had been unable to jump but its condition progressed to tetraparesis, most markedly on the right, during the first 24 hours following onset.

The cat also became dysuric, with a large nonpainful bladder present from time of onset of paraparesis. There was no possibility of trauma or toxic exposure, and the cat did not appear to be in pain. On physical examination, the cat was overweight (body score, 6/9) but other findings were unremarkable. A neurologic examination was performed.

Neurologic examination

Observation

Mental	Alert	X	Depressed		Disoriented		Stupor		Coma	
Posture	Normal		Head tilt		Tremor		Falling	X		
Gait	Normal		Ataxia	X	Pelvic limbs		All 4	X	Circling	
Paresis	Pelvic limbs		Tetra	X	Hemi		Mono			
Other	Nonambulatory tetraparesis that was most pronounced in the right thoracic limb, which appeared flaccid with poor carpal tone and supination; palmigrade stance on the left thoracic limb; and increased muscle tone in the pelvic limbs.									

Postural reactions

Key: 4=exaggerated, clonus; 3=exaggerated; 2=normal; 1=diminished; 0=none; NE=not evaluated

	LF	RF	LR	RR
Wheelbarrow	NE	NE		
Hopping	2	0	1	0
Ext postural thrust			0	0
Proprioceptive pos	2	0	1	1
Hemistand/walk	1	0	1	0
Placing–tactile	2	0		
Placing–visual	2	0		

Spinal reflexes

	LF	RF	LR	RR
Quadriceps			3	3
Extensor carpi	2	1		
Flexion	2	1	3	3
Crossed extensor	0	0	3	0
Perineal			2	2

Cranial nerves

	L	R		L	R	Comments CN
II, VII–Vision menace	2	2	VIII–Nystagmus, resting	0	0	Right-sided miosis, ptosis of the eyelid, and enophthalmos were present.
II, III–Pupils resting	2	3	VIII–Nystagmus, change	0	0	
Stim L	2	2	V–Sensation	2	2	
Stim R	2	2	VII–Facial mm	2	2	
II–Fundus	2	2	V, VII–Palpebral flex	2	2	
III, IV, VI–Strabismus, resting	0	0	IX, X–Gag	2	2	
III, IV, VI, VIII–Strabismus, position	0	0	XII–Tongue	2	2	

Sensation (Locate and describe abnormal)

Hyperesthesia	2	
Superficial pain	2	
Cutaneous reflex	2	
Deep pain	2	

What is the problem? Where is the lesion? What are the most probable causes of this problem? What is your plan to establish a diagnosis? Please turn the page.

Assessment

Anatomic diagnosis

Problem	Rule out location
Tetraparesis (most pronounced in the right thoracic limb) with reduced muscle tone and reduced spinal reflexes in the right thoracic limb; increased spinal reflexes in the pelvic limbs bilaterally	Right-sided lesion within the C6-T2 spinal cord segments
Right-sided miosis, ptosis of the third eyelid and enophthalmos, suggestive of Horner's syndrome	Rostral midbrain and hypothalamus, C1-T3 spinal cord segments, T1-T3 nerve roots, or middle ear

Likely location of one lesion

Because of the combination of the lower motor neuron signs for the right thoracic limb with Horner's syndrome in the right eye, a lesion of the C6-T2 spinal cord segments was most likely.

Etiologic diagnosis—Differential diagnoses considered for the cat of the present report included infectious disease (viral, parasitic, fungal, or bacterial), inflammatory disease, neoplasia associated with the cervical portion of the vertebral column, vascular abnormality (eg, infarction, fibrocartilaginous embolism [FCE], or hemorrhage), and trauma (disk herniation, fracture, or luxation). Plans for diagnostic testing included clinicopathologic evaluation and assessment for serum antibodies against *Toxoplasma gondii*, FIV, FeLV, and *Cryptococcus* spp (to evaluate for infection); CSF analysis (to evaluate for infection, inflammatory disease, or neoplasia in the CNS); abdominal ultrasonography (to evaluate for evidence of systemic disease); MRI of the cervical portion of the vertebral column (to assess for inflammation, neoplasia, vascular problems, or evidence of trauma); and radiography or CT of the vertebral column if a fracture was suspected on the basis of other findings.

Diagnostic test findings—Results of clinicopathologic analyses indicated that the cat had mild anemia (Hct, 24%; reference interval, 25% to 45%) and moderately high alanine aminotransferase activity (141 U/L; reference interval, 15 to 45 U/L). A sample of CSF was collected from the lumbar region, the analysis of which revealed neutrophilic pleocytosis (total nucleated cell count, 213 cells/ μ L [reference interval, < 8 cells/ μ L], of which 94% were neutrophils, 3% were lymphocytes, and 3% were large mononuclear cells). The volume of CSF collected was insufficient for protein measurement. Results of screening for infectious diseases were negative, and serum antibodies against *T gondii*, FIV, FeLV, and *Cryptococcus* spp were not detected.

Findings of abdominal ultrasonography were unremarkable. The cat was anesthetized, and MRI^a of the cervical portion of the spinal cord was performed to obtain T1- and T2-weighted images in sagittal and transverse planes. The T1-weighted images were obtained before and after IV administration of gadopentetate dimeglumine^b (0.1 mmol/kg [0.045 mmol/lb]). The sagittal T2-weighted image revealed an ill-defined hyperintense signal within the spinal cord parenchyma. The affected area was located in the dorsal aspect of the spinal cord; it extended from vertebrae C4 to T3 and was centered around C5 (likely corresponding to spinal cord segments C5 to T3). The hyperintensity was lateralized dorsally to the right side

of the spinal cord, with no obvious mass effect. The lesion was not visible on other images and was not enhanced following IV injection of contrast agent. The normal hyperintense signals of the C7, T1, and T2 intervertebral disks were not visible, and the signal of the C6-7 disk was reduced.

From the cat's clinical signs, especially the sudden-onset lateralized paraparesis, and the MRI findings, the likely remaining diagnoses were FCE with resulting infarction to the spinal cord; a high-velocity, low-volume intervertebral disk extrusion (eg, the C6-7 disk); meningomyelitis (most likely immune mediated); and lymphoma (given that it has been reported that 2/11 cats with spinal lymphoma had a CSF neutrophilic pleocytosis¹). On the basis of the differential diagnoses of lymphoma or meningomyelitis and the advanced neurologic deficits of the cat, treatment with L-asparaginase (400 U/kg [181.8 U/lb], SC, once), prednisolone (40 mg/m², PO, once daily), and lomustine (60 mg/m², PO, once every 21 days) was initiated. The clinical opinion was that this treatment was unlikely to be detrimental in the face of an FCE or high-velocity, low-volume intervertebral disk extrusion. Supportive care was provided (bladder management and prevention of pressure sores). However, the cat developed signs of feline influenza in the week that the treatment was started, and treatment was halted. Gradual improvement in the cat's condition was noted at this stage, with improved motor function of all 4 limbs and resolution of Horner's syndrome.

The cat was returned for follow-up evaluation after 7 weeks and was found to have made a marked recovery. It was able to ambulate outdoors and use the stairs and a litter tray without assistance and was urinary continent. Upper motor neuron signs were present in the pelvic limbs, as well as lower motor neuron signs in the right thoracic limb, but there was no evidence of Horner's syndrome. The cat continued to improve over a further 8-month follow-up period and had only mild neurological deficits of the right thoracic limb. From the week of discharge from the hospital, the cat did not receive any medication, leading to the presumptive diagnosis of FCE.

Comments

Fibrocartilaginous embolism, a form of ischemic myelopathy, is a condition regarded generally as an

uncommon cause of spinal cord lesions in cats.^{2,3} The route of entry of fibrocartilaginous material into the spinal cord vasculature is unknown; however, theories such as the movement of disk material into the spinal arteries or neovascularization between the cord and disk with resultant embolization of material have been proposed.² Ischemic myelopathy may also develop as a result of other embolic disorders (thrombosis, sepsis, parasitism, surgery, or trauma) and vasculitis. Histologic examination of affected tissues is required to definitively confirm the diagnosis.³ Another possible cause of sudden-onset myelopathy in cats, as in dogs, is high-velocity disk extrusion myelopathy; however, this association has not been definitively proven on the basis of histopathologic findings in affected tissue in cats.⁴⁻⁶

In clinical cases, FCE is characterized by neurologic signs of an asymmetric myelopathy, usually in middle-aged to older cats.^{2,3,7,8} Initial clinical signs can vary depending on the spinal cord segments affected (cervical vs thoracolumbar). In cats, FCE injury most commonly occurs at the cervicothoracic intumescence.^{2,3,7-9} In FCE-affected cats for which a CSF sample has been analyzed, neutrophilic pleocytosis is typically reported,^{2,9} although no CSF abnormalities have been described for 1 affected cat.³ There are only reports of 3 cats with suspected disk extrusion myelopathy, and for each of those cats, the lesion affected the cervical, thoracic, or lumbar portion of the spinal cord.⁴⁻⁶ Among those 3 cats, a CSF sample was collected from 1; analysis revealed neutrophilic pleocytosis.⁵

In the case described in the present report, tetraparesis, lower motor neuron signs for the right thoracic limb, and right-sided Horner's syndrome made up an interesting combination of deficits that helped to localize the origin of the disease to the C6-T2 region of the spinal cord, as confirmed via MRI. Lower motor neuron signs are usually highly informative because they allow a clinician to narrow the neuroanatomic localization of a lesion to a region smaller than that indicated by upper motor neuron signs.

The Horner's syndrome in the cat of this report most likely resulted from a lesion on the right side of the spinal cord at the level of the T1-T3 spinal cord segments, where the first-order neurons of the sympathetic innervation to the eye synapse with the second-order neurons. Horner's syndrome as a result of FCE to the C6-T2 region in cats has been previously reported.^{2,9}

It is difficult to provide a prognosis for cats with FCE since it is a relatively newly recognized spinal cord disease in this species. With or without treatment, prognosis is considered guarded to good, depending on the localization of the lesion and initial neurologic signs; for cats in which the cervical region is affected, the prognosis is thought to be favorable. In general, there is no treatment other than supportive measures for the autonomic system (respiratory and urinary functions). Half of cats described in the earlier reports^{2,7,9} were euthanized, which might have been because of a lack of awareness of the disease; however, in a more recent study,³ all 6 cats survived for at least 20 months

following diagnosis, including 1 that had no response to noxious stimulation at the initial evaluation. In all 3 cases of suspected disk extrusion myelopathy, neurologic function was recovered with supportive care.⁴⁻⁶

It is possible to make a parallel between FCE and diseases or trauma causing contusion and ischemia to the spinal cord, such as high-velocity disk herniation or road traffic accidents. Dogs with FCE of the cervical portion of the spinal cord usually have a better prognosis than those with damage at the cervicothoracic or thoracolumbar intumescence or those with loss of pain sensation, possibly because a complete cervical lesion would likely result in death due to diaphragmatic paralysis (innervation arises from cervical nerves C3 to C5) and subsequent asphyxiation prior to initial evaluation. In cats, factors regarding prognosis are less clear, with improvement of clinical signs having been reported for 1 cat with loss of pain sensation.³

- a. Philips Gyroscan NT Intera (1.5 T), Philips Healthcare, Guildford, Surrey, England.
- b. Magnevist, Bayer Plc, Newbury, Berkshire, England.

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This feature is published in coordination with the American College of Veterinary Internal Medicine on behalf of the specialty of neurology. Contributors to this feature should contact Dr. Helen L. Simons (800-248-2862, ext 6692) for case submission forms. Submissions will be sent to Dr. Karen Kline, DVM, DACVIM, for her review, except when Dr. Kline is an author.